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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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Albers-Schönberg Disease. With Report of Two Cases in An Egyptian Family.
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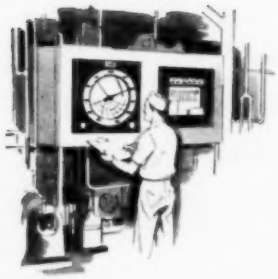

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* Shwachman, H., Children's Hospital, Boston, The Child in Health and Disease, Grulee and Eley, 2nd Edit.

Butler, A., Harvard Medical School, private communication.

Royce, S., Los Angeles Hospital, private communication.

Clay, W. A., Frank, D. W., and Logan, V. W., Studies on the Therapy of Pancreatic Insufficiency, Gastroenterology, 26, 2, (1954).

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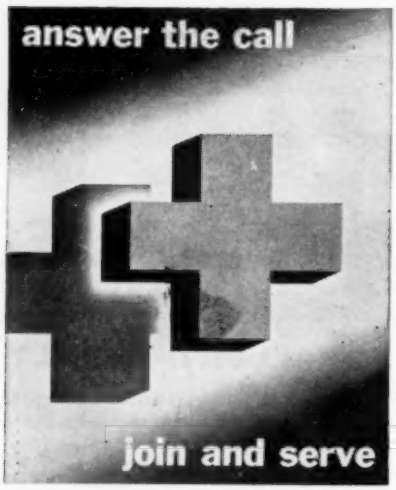

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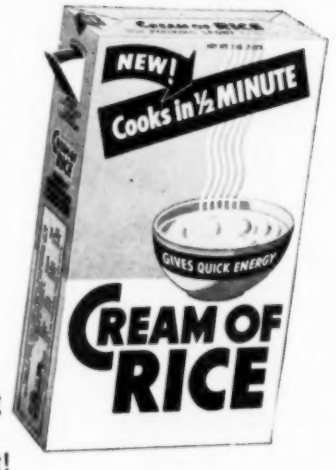

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References: 1. American Heart Association: Mod. Concepts Cardiovas. Dis. *22*:160 (Jan.) 1953. 2. Stollerman, G. H.; and Rusoff, J. H.: J.A.M.A. *150*:1571 (Dec. 20) 1952

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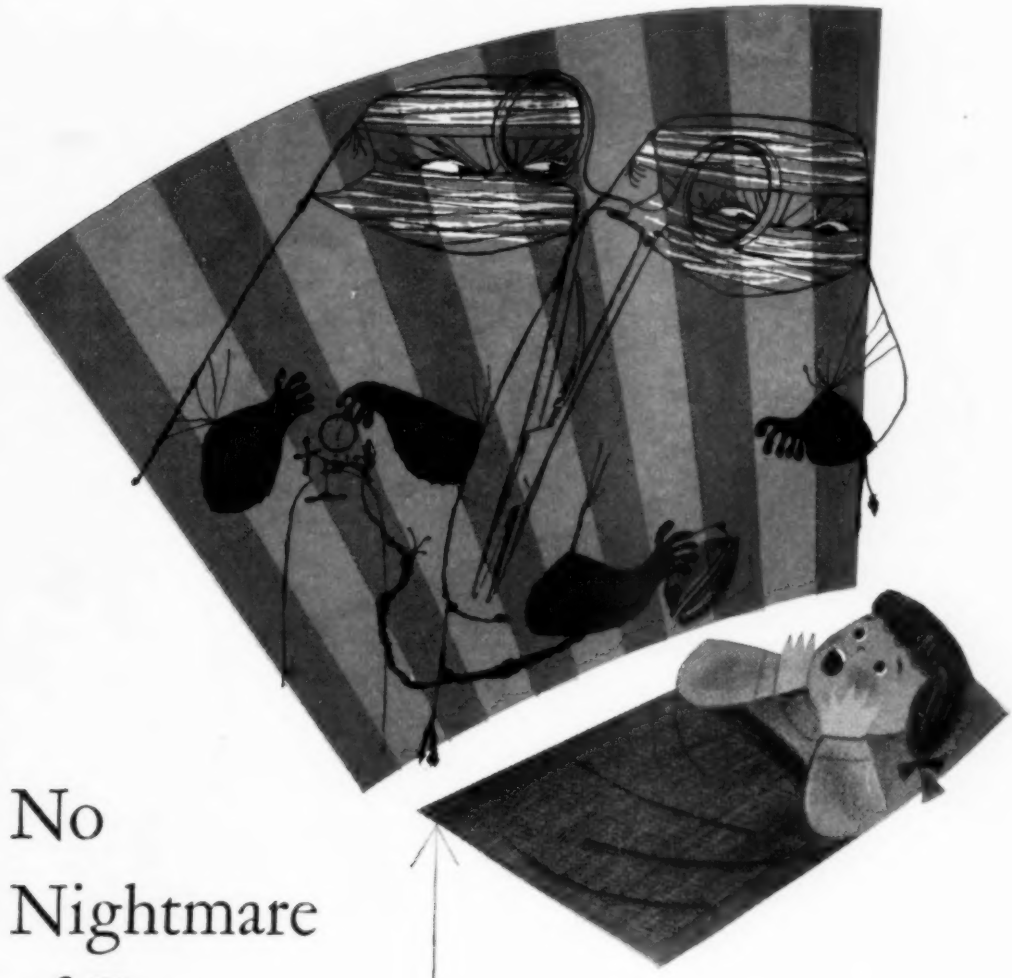

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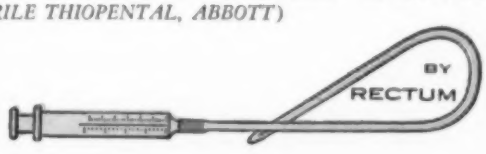

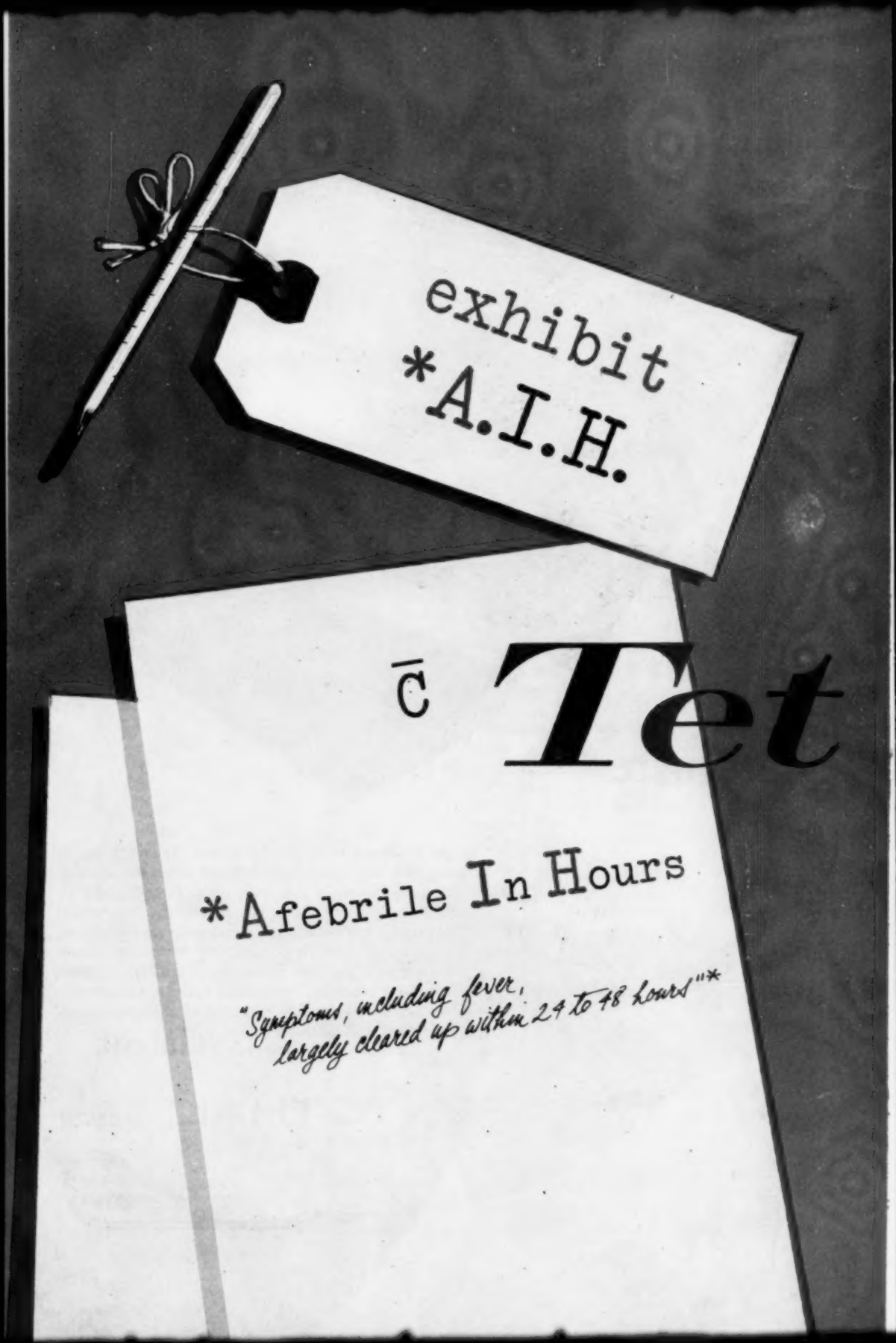
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**English, A. R., et al.: Antibiotics Annual (1953-1954), New York, Medical Encyclopedia, Inc., 1953, p. 70.*

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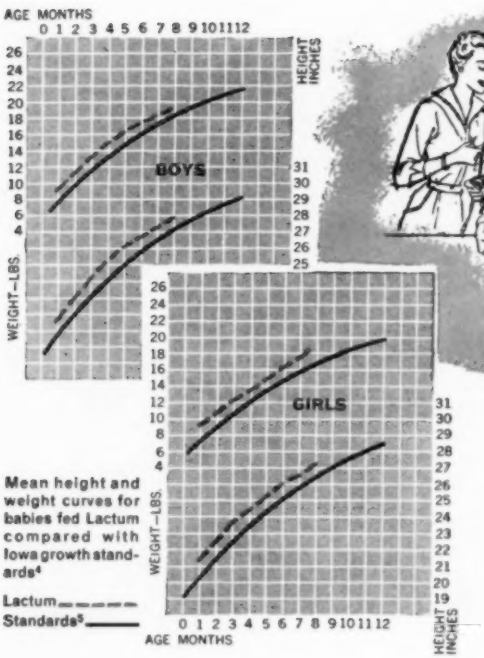
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1. Jeans, P. C., in A.M.A. Handbook of Nutrition, ed. 2, Philadelphia, Blakiston, 1951, p. 275; 2. Albanese, A. A.: Pediatrics 8: 455, 1951; 3. Holt, L. E., Jr., and McIntosh, R., in Holt's Pediatrics, ed. 12, New York, Appleton-Century-Crofts, Inc., 1953, pp. 173-178; 4. Frost, L. H., and Jackson, R. L.: J. Pediat. 39: 585, 1951; 5. Jackson, R. L., and Kelly, H. G.: J. Pediat. 27: 215, 1945.

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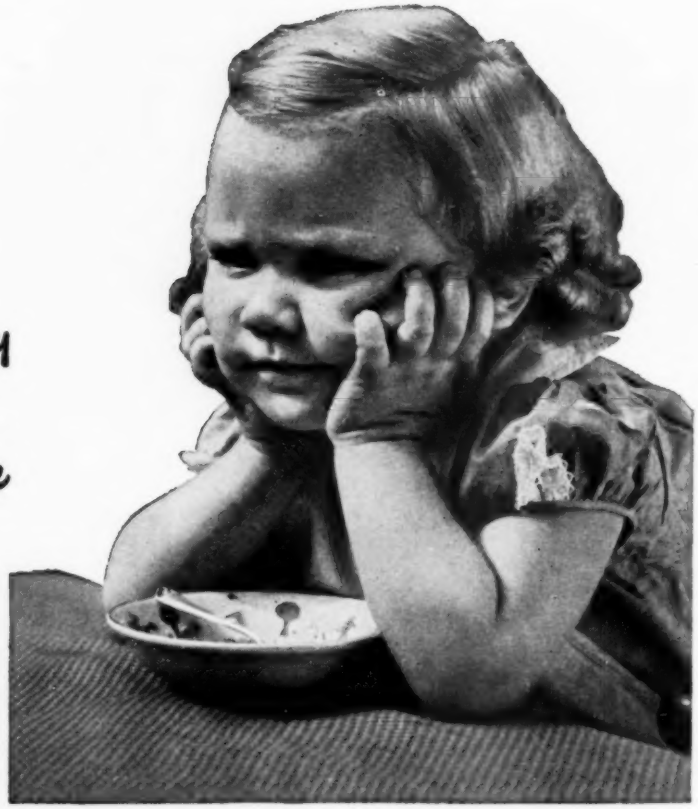

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FOR: Peri-Anal Dermatitis

CRITERIA: Inflammation centered around the anus from 3 to 4 cms. in diameter and frequent stools.

CAUSE: Transitional stools in the newborn, diarrhea or following oral antibiotics.[1]

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1. Manheim, S. D., et al: "Further Observations on Anorectal Complications Following Aureomycin, Terramycin and Chloromycetin Therapy." N. Y. State Jrnl. Med., 54:37-1, Jan., 1954.
2. Curry, J. C. and Barber, F. W.: Bacteriological Proceedings, 1951, of The Society of Am. Bact., page 23.
3. Grossman, L., St. Francis Hospital, Miami Beach, Fla., to be published.
4. Niedelman, M. L., et al: Jrnl. Ped., 37:762, Nov., 1950.
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6. Benson, R. A., et al: Jrnl. Ped., 31:369, Oct., 1947.
7. Ibid: Jrnl. Ped., 34:49, Jan., 1949.

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ARCHIVES OF PEDIATRICS

VOL. 71 MAY 1954 No. 5

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ALBERS-SCHÖNBERG DISEASE*

WITH REPORT OF TWO CASES IN AN EGYPTIAN FAMILY

M. A. ABBOUD, M.R.C.P.,D.C.H.,
Z. H. ABDIN, M.R.C.P.,D.Ch.
AND
O. ALFY, M.D., D.Ch.
Cairo, Egypt.

Albers-Schönberg disease is a relatively rare bone disease. In the 10 years following its discovery by Albers-Schönberg[1], in 1904, no other cases were reported. In 1934, McCune and Bradley[2] collected 69 cases from the literature. By 1941, the number of reported cases amounted to 135 (Higinbotham and Alexander[3]). In 1947, Pines and Lederer[4] raised the number to 148, and by 1950, it reached 174 (Kneal and Sante[5]). In this country we could not come across a case in children or in adults. The reported family shows the existence of the disease in Egypt, and, as the disease has a definite herdofamilial tendency[6], it is quite probable that more cases may be met with in the future on careful inquiry.

CASE REPORTS

Case 1. F. A. H., a girl 8 years old, was admitted to hospital for cough and expectoration of three years' duration. Apart from delayed teething and starting to walk at three years—in spite of giving numerous vitamin D injections—the past history was irrelevent. Examination showed a dwarfed girl (height 103 cms.) with a somewhat shorter span (95 cms.). The oblong vault com-

*From the Pediatric Department, Kasr El-Ainy Hospital, Cairo, Egypt.

pared to a relatively small face was rather striking. The anterior and posterior fontanelles, as well as the coronal, sagittal and lambdoid sutures, were widely open. The skull circumference was 51.8 cms., the teeth were crowded but not decayed, and the eyeballs were slightly proptosed. A localized swelling of the left leg was detected, over the middle of the tibia, overlying the fracture, which was demonstrated on radiological examination. The hands looked rather small. The mentality, hearing and vision were normal. The skin was smooth, the hair glistening, greasy and abundant. The lungs showed few consonating crepitations over both bases, more on the left side. No enlargement of the liver, spleen or lymph glands. Blood pressure 95/60.

Radiography showed marked sclerosis of all the bones of the body, especially in the femora (Fig. 1), tibiae (Fig. 2), spine and pelvis (Fig. 3), radius and ulna (Fig. 4). The medullary cavity was markedly reduced, and almost obliterated in the bones of the forearm. The cancellous bone at the metaphysis showed coarse trabeculation, especially at the lower end of the femur and both ends of the tibia. In the epiphysis, the trabeculation gave the appearance of a dense fine meshwork. There was widening of the lower end of femur. The lower surface of the neck of the femur showed fraying and fragmentation, possibly from narrowing of the nutrient canals by the sclerosing process[5]. No coxa vara or valga. The ribs showed marked thickening of the cortex, which was well demonstrated on either side of the thoracic cage in the postero-anterior view. The skull (Fig. 5 and 6) showed marked scelerosis and wide patency of the two fontanelles, coronal, sagittal and lambdoid sutures. The posterior clinoid processes were thickened and bulbous, but with no deformity of the pituitary fossa. The

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LEGENDS FOR CUTS ON OPPOSITE PAGE

Fig. 1. Femora, showing marked sclerosis and thickening of the cortex.

Fig. 2. Tibiae, showing sclerosis and thickening at the middle of the shafts at the site of the healing fractures.

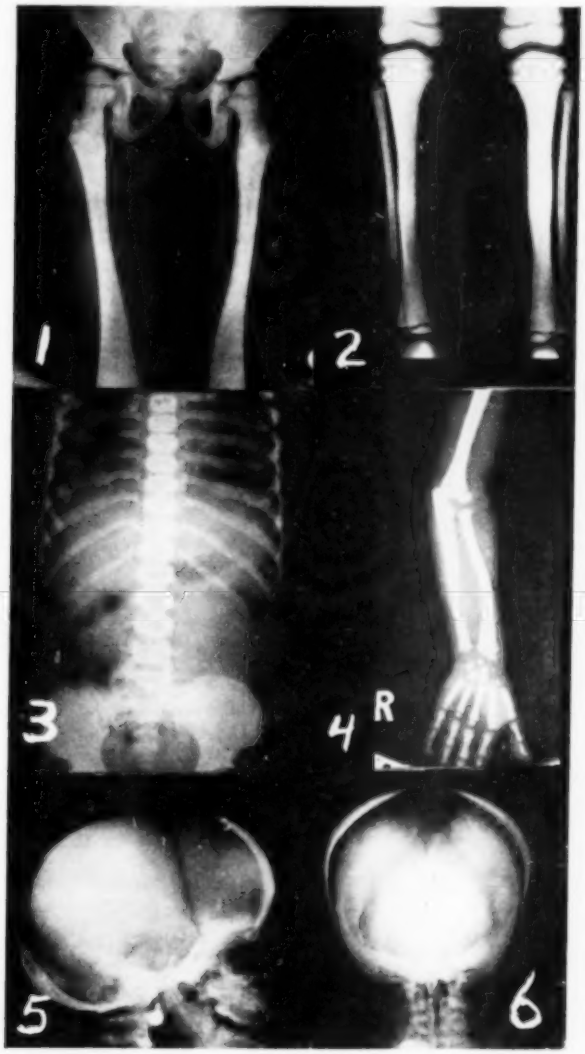
Fig. 3. Ribs, spine and pelvis, showing marked sclerosis.

Fig. 4. Lower end of humerus and bones of the forearm and hand. The medullary cavity is almost completely obliterated in the ulna.

Fig. 5. Lateral view of the skull showing patency of both fontanelles, and coronal and lambdoid sutures.

Fig. 6. Postero-anterior view of the skull showing patency of the sagittal suture and the marked sclerosis.

angle between the body and the ascending ramus of the mandible was markedly obtuse. Both tibiae showed partially healed transverse fractures in the middle of their shafts. There was no ap-



preciable progress in the process of healing of these fractures on radiographic examination, six months later. Radiography of chest normal.

Blood examination showed: hemoglobin 82 per cent, R.B.C. 4.6 million per mm.^{3}, W.B.C. 17.2 thousand per mm.^{3} (polymorphs 49 per cent, lymphocytes 47 per cent, eosinophils 4 per cent). No immature white or red cells were detected. Serum alkaline phosphatase normal (11 King-Armstrong units), serum calcium 9.2 mgms. %, blood phosphorus 3.2 mgms. %, urea 32 mgms. % and cholesterol 188 mgms. %. Wassermann reaction, negative. Alkalie reserve 67.2 vol. CO_2 %. Cerebrospinal fluid tension was 230 mms. water, cell count 5 per mm^3., proteins 10 mgms. %, chlorides 738 mgms. %, and sugar 55 mgms. %. No changes were seen in the cornea by slit lamp examination. Examination of the urine showed normal findings, with normal amount of calcium excretion.

Fig. 7. Face of boy showing the depression at site of coronal sutures, bulging forehead and the slight proptosis.

Fig. 8. Laternal view of face showing bulging forehead and peaked nose.

Repeated trials for bone marrow puncture failed due to the sclerosis.

A survey of the family of the first case revealed a younger brother similarly affected.

Case 2. M. A. H., a boy 5 years old, apparently in good health. He was somewhat shorter than is expected for the age—height 96 cms., span 90 cms., distance from vertex to umbilicus 48 cms. He cut his first tooth and started to walk by his first birthday. On examination, the anterior and posterior fontanelles were both widely patent (anterior fontanelle 5 cms. in diameter and the posterior 2 cms.). A pronounced depression was seen at the site of

the coronal suture (Fig. 7), which was also patent. Skull circumference 50.6 cms. The forehead was bulging (Fig. 8), eyeballs slightly proptosed, and nose peaked. The fingers were rather short. Mentality, hearing and vision normal; skin smooth, hair glistening and abundant. Heart and lungs free, no enlargement of liver, spleen or lymph glands. Blood pressure 95/65.

Radiology showed similar, but even more pronounced, changes as in his sister (Case 1). Skull changes were also similar in both cases. The posterior clinoid processes were enlarged and bulbous, and the pituitary fossa was distorted, with antero-posterior diameter markedly prolonged (18 mms.).

Blood examination showed hemoglobin 100 per cent, R.B.C. 5 million per $mm.^3$, W.B.C. 9.6 thousand per $mm.^3$ (polymorphs 38 per cent, lymphocytes 54 per cent, monocytes 2 per cent, eosinophils 6 per cent). No immature white or red cells were detected in the peripheral circulation. Bone marrow examination showed a hypocellular picture with the erythroid-myeloid ratio of 1/6.5. Lymphocytes 25 per cent, erythroid series 10 per cent and myeloid series 65 per cent (myelocytes 30 per cent, metamyelocytes 15 per cent and segmented 20 per cent). Marked resistance was encountered during the bone marrow puncture due to the scelerosis.

DISCUSSION

Reviewing the cases described, the disease is found to affect both sexes almost equally with a slight prevalence in males. Most of the cases were in childhood, though a good number were in infancy, and few cases were reported even in utero (Clifton et al.[7]; Jenkinson et al.[8]). Pirie showed changes in the bones of a fetus whose mother suffered from osteopetrosis; the development of the baby was followed by him for six years. Some cases were described in adolescence, and very few in old age; the oldest case reported was 75 years (Fairbank[9]). Albers-Schönberg's case was aged 26. Whether or not the disease can be acquired in adult life is questionable.

The etiology and pathogenesis of the disease is still obscure. An heredofamilial tendency exists, and no race is exempt. Consanguinity was occasionally found to have a definite role. An endocrine basis for the disease has been suggested, and in one of

the cases an enlarged parathyroid gland was described, but endocrine therapy seems to be of no avail. Some of the reported cases were complicated by Hodgkin's disease (Herscher and Stein[10]), or osteogenic sarcoma (Kerr[11]). Some cases were associated with manifestations of syphilis. Others occurred in conjunction with rickets (Karshner[12]; Kramer and Helperl[13]); in such cases, in addition to the rachitic epiphyseal changes, a heavy periosteal reaction is seen investing the shaft of the bones. Fluorine poisoning has been considered an etiologic factor (Speder[14]), owing to the similar chalky appearance of the bones in both conditions; but this seems unlikely as calcium fluoride crystals were not observed in the Haversian systems in any of the cases.

The great excess of lime salts in the bones, together with the gradual encroachment of this sclerotic bone on the medullary cavity is responsible for almost all of the clinical findings. Interference with the bone marrow results in myelophthisic anemia[15], which is the underlying cause of the constitutional manifestations, and of the occasional curtailed longevity. The hematologic findings were thoroughly reviewed by McCune and Bradley[2]. Wide variations in the blood picture occur in different cases, depending on the extent of bone marrow affection, though in some cases the extent of marrow affection and anemia do not seem to run parallel. Extramedullary hematopoiesis may occur in the liver and spleen, causing their enlargement, especially in infants. Sometimes no anemia develops at all. Other clinical manifestations, which may occur, are retarded growth, hydrocephalic changes, optic atrophy, irregular thickening of the skull, calcification in the ligaments and dural folds, and imperfect dentition. Despite the sclerotic changes, the bones are usually fragile, and transverse fractures are of common occurrence. Serum calcium and blood phosphorus are usually normal, but the phosphatase may be decreased. Some cases were reported in which serum calcium was elevated to double its normal value (Schulze[16]). Urinary changes are insignificant. Blood pressure is usually normal, though it may be elevated late in the disease.

Roentgenologically, the manifestations may be described as those of the infantile or growing stage, and those of adult or established stage. In the former, the changes may appear in the fetus in

utero[7, 8], or a few months or years after birth (Fairbank[17]). The metaphyseal end of the diaphysis becomes dense, and the condition may be mistaken for healing rickets. This sclerosis progressively increases in density, and extends into the shaft. The cancellous regions at the ends of long bones may be widened and show parallel longitudinal striations. In adults, the bones show a more or less homogeneous structureless dense appearance.

Partial cases affecting one or several bones have been described. Wakely[18] reported a case showing patchy distribution of the dense areas in the bones.

Sclerosis of the skull bones may encroach on the various orifices in the skull, and is responsible for the optic atrophy or nerve deafness which may occur. Clubbed enlargement of the posterior clinoid processes may press on the pituitary gland, and is said to be a factor in the tendency to dwarfism which may occur[5] (Case 1).

Spontaneous remissions may occur for some period[17], followed by relapses, and this alternation of remissions and relapses may explain the occasional appearance at the ends of long bones of dense transverse bands, alternating with bands of lesser, or normal, density. Deformities, as coxa vara and genu valgum, are of common occurrence. Fractures are frequently seen in long bones. They are usually transverse. They may not cause any disability to the patient, in which case they are only detected radiologically (Case 1). Healing of these fractures may occur rapidly, or may be delayed for more than a year (Alexander[19]).

No treatment is found effective for this disease. Vitamin D depletion, or parathormone administration failed to produce any effect.

SUMMARY

Two cases of Albers-Schönberg disease are reported in an Egyptian family.

The first case was admitted with a pulmonary complaint, was dwarfed but showed no abnormality of the pituitary fossa.

The second case was spotted during a survey of the family, showed little if any dwarfism, but marked abnormality of the pituitary fossa.

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IS HUMAN POLIOMYELITIS CAUSED BY AN EXOGENOUS VIRUS?

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(*Continued from April issue*)

INCUBATION PERIOD OF HUMAN POLIOMELITIS VERSUS EXPERIMENTAL ANIMAL POLIOMYELITIS

Agreement regarding the incubation period of cases of human poliomyelitis has been based almost entirely on the results of animal experiments with the virus of the disease. It is a well known fact that cases of human poliomyelitis in a home, institution or community occur almost simultaneously and are often described as explosive in character. This fact is typical of the effects of poisoning. On the other hand, where experiments on animals in the laboratory are carried out with the virus, a definite incubation period can be established according to the manner in which the virus is administered, its concentration, and the species of animal that is employed. It has always been difficult to reconcile the fact that human poliomyelitis has a short incubation period of one to three days, according to Wickman[55] and others, where the virus would necessarily have to traverse the natural barriers in order to set up infection in the central nervous system and an incubation period of as long as nine or more days in the experimental disease, where the virus is inoculated directly into the central nervous system.

Multiple cases in families present the nearest approach to the grouping of epidemiologically connected cases. There is no conclusive proof that the disease spreads under such circumstances like a contagious or infectious disease. The cases in these families occur simultaneously as in cases of poisoning. Aycock and Eaton[71] (1925) collected the records of 576 multple cases in 253 families. They found that the proportion of secondary cases is highest on the day of the appearance of the first case, and the proportion tends to diminish steadily as time elapses.

PRECIPITATORS OF HUMAN POLIOMYELITIS

Certain factors appear to be necessary in many cases for precipitating the manifestations of human poliomyelitis. These include: fatigue, chilling, trauma, heat and humidity, operative procedures, or pregnancy. Similar factors are well known precipitators of the manifestations of lead toxicity. Neurological manifestations of lead poisoning, and presumably other poisons, occur more frequently in hot weather than during any other season of the year. Suzuki and Kanako[72] (1924), Fukushima and Matsumato[73] (1928), Blackman[74] (1937), Rappaport and Rubin[75] (1941), and Guannattasio et al.[76] (1951) have all pointed out this fact. It is well known that the neurological manifestations of alcohol poisoning (delirium tremens) can be precipitated by overexertion, exposure, operations, trauma, shock, fright and acute inflammatory disease.

COMPARATIVE PATHOLOGY OF HUMAN VERSUS EXPERIMENTAL ANIMAL POLIOMYELITIS

The differences between human poliomyelitis and the experimental animal disease is definitely shown by the divergence of the pathological lesions in the two diseases. It must be admitted that the pathological lesions in the nervous system of experimental animal poliomyelitis are similar to, if not identical with, those in cases of human poliomyelitis. However, the visceral lesions that occur in cases of human poliomyelitis cannot be reproduced in the experimental animal with the virus regardless of the manner of administration or its concentration. Thus, there are to be found in human poliomyelitis many evidences of pathology besides the presence of a virus and viral reaction. In the stomach there is a very high incidence of submucosal petechial hemorrhage associated with intense congestion of the mucosa. Myocarditis, accompanied by mycardial degeneration, has been found in a high percentage of cases. In the parenchymatous organs, especially the liver and kidneys, there is usually demonstrated degeneration of the sort usually described as cloudy swelling. Landon and Smith[77] (1934)pointed out that the granular degeneration of the parenchymatous cells, as well as fatty degeneration represent toxic changes. They state also that the kidneys in cases of human poliomyelitis

show changes that may be attributed to a general systemic toxemia. In practically all cases of human poliomyelitis there is intense congestion of the blood vessels throughout the body.

The widespread lymphoid hyperplasia found consistently in gross and microscopic autopsy examinations in cases of human poliomyelitis never has been explained on the basis of a virus infection. There is involvement of Peyer's patches and the solitary follicles of the gastro-intestinal tract, mesenteric and retroperitoneal lymph nodes, peribronchial lymph nodes, thymus, malpighian corpuscles of the spleen, tonsils, adenoid tissue of the nose and throat, and the lymph nodes of the neck, axilla, groin, and other parts. Burrows[78] (1931) in a series of about fifty autopsies, noted that the maximum amount of lymphoid hyperplasia was in Peyer's patches and the solitary lymph follicles of the gastro-intestinal tract and the mesenteric lymph nodes. He felt that the nerve tissue changes were secondary to those existing in the lymph channels of this tissue. It is a well known fact that lymphoid hyperplasia can occur as a result of poisons and toxins. Doubtless the lymphoid hyperplasia in human poliomyelitis is an expression on the part of the body to poisons and toxins in which protection is afforded by hyperplasia of its reserve forces, the lymphatic apparatus.

The intense reaction in the gastro-intestinal tract not only explains the reaction to a poison but likewise the clinical manifestations of gastro-intestinal irritation that so frequently occur in cases of human poliomyelitis. Although many workers have postulated that the gastro-intestinal tract is the portal of entry of the poliomyelitis virus into the human body, this never has been proven conclusively. Numerous investigators have failed to infect monkeys when the virus has been administered orally even in high concentration and in no case have the gastro-intestinal tract lesions of human poliomyelitis been duplicated in experimental animals. The virus that is recovered from the feces of human poliomyelitis is probably synthesized or activated within the gastro-intestinal tract and is excreted therefrom without gaining entrance to the body. This conclusion appears to be justified by the failure to infect animals orally, and by the work of Gard[79] of Sweden. Gard's extensive studies suggested that intestinal protein is an avirulent or non-neurotropic varient of the poliomyelitis virus, a normal

inhabitant of the intestines. This intestinal protein (virus), according to Gard, is non-pathogenic, but under the influence of exogenous factors is pathogenic when injected into experimental animals. Further proof that the virus in the intestinal tract does not enter the human body to cause human poliomyelitis is indicated by repeated failures to isolate a virus from the blood stream, although human poliomyelitis is generally considered to be a generalized systemic infection. The isolation of a virus from the blood stream would not necessarily indicate that it gained access to the body from the external environment.

ANTIBODY FORMATION AND HUMAN POLIOMYELITIS

There has been much controversy regarding the interpretation of the presence of poliomyelitis virus neutralizing antibodies in human sera. It is now assumed that these antibodies develop following non-paralytic and probably non-symptomatic infection with the virus during childhood as well as in frank cases of paralytic poliomyelitis. Some workers have tried to prove the contagiousness of human poliomyelitis by demonstrating elevated antibodies in contacts. Other workers have been unable to find the antibodies increased in contacts[80]. Poliomyelitis can develop in the presence of neutralizing antibodies and many patients convalescent from the disease show no antibody. The serums of guinea-pigs and rabbits do not contain neutralizing antibody for monkey passage virus, though it is well known that these animals possess an absolute immunity to the virus. Further, antibodies are found in a high percentage of natives of lands in which poliomyelitis is unknown and paradoxically in a much smaller percentage of the population where the disease occurs in epidemic form. The extent of natural resistance to human poliomyelitis is utterly disproportionate to the quantities of virus which is postulated to be present in the general population by analogy with the results of experimental animal poliomyelitis.

It is noteworthy that the appearance of neutralizing antibodies in the blood after the injection of the poliomyelitis virus is very uncertain evidence of parallel immunity to the natural disease[81]. This fact was shown clearly by Kramer[82], in 1936. He vaccinated a group of children with vaccine and two months later found that 50 per cent had developed neutralizing antibodies. However, in a

parallel uninoculated group of children, 41 per cent had also developed antibodies. Kramer's results were in essential agreement with those of Aycock and Hudson[83], who found an increase of 28.6 per cent of immunes among the vaccinated children in their series as compared with an increase of 22.8 per cent of immunes in the unvaccinated control group. Neither of these writers considered the small difference of any practical value in favor of the vaccinated group.

KOCH'S POSTULATES AND HUMAN POLIOMYELITIS

Koch's first and one of his most important postulates emphasizes that a parasite must be found in *every* case of the disease in order to state that that particular parasite is the cause of the disease. In 1952, there were nearly 58,000 cases diagnosed as poliomyelitis in the United States, 75 per cent of which were paralytic. Attempts to isolate the virus from the excretions were carried out in little more than one per cent. In the remaining 99 per cent, a virus was merely assumed to have been present. That the paralysis in this large group might have had many causes is obvious. It is not to be overlooked that a significant number of spinal cords removed from children dying in the acute stage of poliomyelitis are incapable of producing paralysis after intracerebral inoculation in the monkey.

Korns[84] (1953), N. Y. State Public Health Officer, states that the isolation of the poliomyelitis virus for diagnostic purposes is almost never done and is of little practical value, while at the same time being a very difficult procedure. He points out further that isolation of the poliomyelitis virus from the patient by no means establishes the diagnosis since the virus is widely prevalent in the population during epidemic periods without producing disease. Bell[85], epidemiologist, National Institutes of Health, states that the isolation of a virus from poliomyelitis patients is not a routine or reportable procedure; it is carried out only in conjunction with research studies. Serological tests of acute and convalescent blood specimens, he says, are often of no value for determining the cause of the current infection in poliomyelitis because in this disease the antibodies are usually in good titer at the time of onset of symptoms.

BIOCHEMICAL FACTORS IN HUMAN POLIOMYELITIS

There appear to be certain physiological and chemical factors which seem to be necessary for the development of human poliomyelitis and for the synthesis or activation of the virus. Hormonal imbalance has been suggested as an important factor by a number of writers in predisposing an individual to poliomyelitis. Stern[86] (1911) found that a considerable number of cases of this disease showed some symptoms of Basedow's disease, i.e., goiter, tachycardia, tremor, nervousness, etc. Wynkoop[87] (1916) suggested that human poliomyelitis is a disease caused by negation of glandular efficiency. Draper[88] (1932) noted signs in his patients that tended to point toward endocrine deficiencies. The mothers of these poliomyelitis patients not infrequently had moderate or marked exophthalmos or thyroid enlargement. Inglessi[89] (1932) found hypocholesterolemia in thirty children with poliomyelitis during the acute stage and for some time in the paralytic stage. Jungeblut[90] (1932) suggested that the mass protection enjoyed by the adult human population rests primarily on the normal function of the endocrine balance characteristic of mature age. Later[91] (1935) he stated that the protective action against poliomyelitis probably lies in the normal physiological function of the organism and that the main cause for susceptibility is a hormonal dysfunction. Prophylaxis he suggests must consist mainly in correcting the individual susceptibility on a general physiologic-hygienic basis. Aycock[92] (1940) stated that the susceptibility to poliomyelitis is determined by an inherent endocrinopathy which is largely subclinical. Aycock[93] (1940) found a higher average excretion of estrogenic substance in a group of poliomyelitis patients from one to twenty years after an attack of this disease and noted that there was nothing to indicate that this was a sequel of poliomyelitis. This fact suggested to him that endocrinopathy as a basis of susceptibility does not lie in a simple deficiency in the elaboration of estrogenic substance, but rather in some discrepancy in its economy. Aycock pointed out the frequency with which the paralytic disease tends to parallel certain seasonal and climatic fluctuations in physiologic processes. "Such a correlation suggests," he says, "that susceptibility to paralytic poliomyelitis does not lie in any

fixed anatomical character, but is dependent on some physiological process."

The well known fact that virus activity, as well as the reaction to poisons and toxins, produces chromatolysis in an affected nerve cell indicates the necessity for knowledge of the localization of materials and chemical reactions within the cell. Chromatolysis suggests a shift in the balance of a steady state by differential inhibition or acceleration of complex enzyme-regulated reactions[94]. "In addition to the specific production of chromatolytic changes by toxins and neurotropic viruses, interference with enzyme mechanisms by homonal imbalances or dietary deficiencies might conceivably in extreme cases produce the phenomenon of chromatolysis."[94] Aycock and Foley[95] (1945) stress the fact that motor neurone disease may be brought about by an enhancing or inhibiting action on one or more of the enzyme systems.

A study of the biochemical changes that arise during the course of human poliomyelitis has not been followed adequately, but a few important clues have been reported. One of these consists of the presence of coproporphyrin III in the urine of poliomyelitis patients[96, 97]; another is the appearance in the blood of increased amounts of guanidine.[98, 99]. It is not to be overlooked that both of these chemicals are present in the body in increased amounts in cases of poisoning by a number of toxic agents.

Kaplan et al.[100] (1938-39) described an increase of proteases in the cerebrospinal fluid of cases of human poliomyelitis. Kovacs[101] found (1953) in this disease that there are no changes of acid soluble inorganic phosphorus resulting from the interaction of enzymes and phosphorus-containing organic material in the cerebrospinal fluid. In acute bacterial meningitides, on the other hand, a great increase of phosphorus was usually evident. Kovacs[102] (1953) studied the nucleases in the cerebrospinal fluid in cases of human poliomyelitis and found consistently high values. His findings suggested some direct connection between chromatolysis and ribonuclease activity.

The fact that ascorbic acid[103-105], thiamin[106-113], methylene blue[114], as well as iodine[115] have been successfully employed by some workers in the treatment of human poliomyelitis, suggests that certain

biochemical disturbances within the body during the course of the disease can be corrected with chemotherapy. In the treatment of a case of bulbar poliomyelitis, Eskwith[116] (1951) postulated that dimercaprol (BAL) might be effective because in heavy metal poisoning it combines with the metals and protects certain enzymes —those containing a sulfhydryl group—from combination with the poison and because viruses seem to cause necrosis by destroying or inhibiting certain intracellular enzyme systems. He reasoned that if glutathione and other sulfhydryl containing enzymes and tissue protein can be injured by heavy metals, it seems quite possible that they can combine with and be injured by other substances besides metals. Similarly, it is quite possible, he thought, that since dimercaprol contains two sulfhydryl groups, it may protect the enzymes from these non-metallic toxic agents. Eskwith's patient was a 4½ year old girl who had required a tracheotomy and oxygen therapy and whose clinical course was steadily downhill until the dimercaprol injections were given. At the end of 24 hours after therapy was begun, the patient was clinically improved and consciousness rapidly followed. Eskwith learned subsequently that some work had been done with dimercaprol in relation to neurotropic virus infections in experimental animals and that there was no evidence of its efficacy. This fact appears to confirm the belief that human poliomyelitis and the artifically produced experimental animal disease are two entirely different entities.

EXPERIMENTAL ANIMAL POLIOMYELITIS IN HUMAN BEINGS

Once the poliomyelitis virus is recovered from human and extrahuman sources many diversified experiments can be carried out in the laboratory with experimental animals. The unfortunate thing, however, is that these laboratory experiments on animals are interpreted as being applicable to the human disease from whence the virus was obtained and that unjustified conclusions are drawn.

Realizing that an animal will develop experimental poliomyelitis from a virus introduced into its body in an abnormal manner, one can expect that a human being also can develop poliomyelitis of the experimental animal type under the same conditions. Thus, there is to be found in the medical literature reports of the development of poliomyelitis in technicians[117-120] working in labora-

tories with concentrated forms of the poliomyelitis virus. In these cases the portal of entry of the virus is doubtless an abrasion, scratch, laceration or needle prick. A case of poliomyelitis in a technician[118], which followed the contamination by a virus of a scratch, failed to show at autopsy the pathological lesions characteristic of human poliomyelitis arising in a natural manner. It is significant that in this case the gastro-intestinal tract revealed no lesions and no virus was present in the intestinal contents. Over the past 40 years these are the only reported cases of poliomyelitis developing in laboratory workers.

If humans are injected with a concentrated form of active virus, it is natural to expect that they would develop the same type of poliomyelitis that occurs in experimental animals following the injection of the virus. Actually, this did occur in 1935, when some children who received a poliomyelitis vaccine prepared with the virus obtained from experimental animals developed poliomyelitis; half of them died[121]. Significant facts of great importance in these cases were that the incubation period of 6 to 14 days following the injections corresponds with the incubation period of experimental animal poliomyelitis; the fact that the level of the spinal cord first affected corresponded with the extremity in which the injection was made, i.e., the same limb or the contralateral limb parallels recent observations. It is now known, for example, that poliomyelitis can occur following the injection of toxic antigens during the summer months, i.e., pertussis vaccine and diphtheria toxoid, and that the paralysis occurs in the same limb or the contralateral limb where the antigen is injected.

COMMENT

The evidence never has been strong nor very convincing that an exogenous virus enters the human body to cause poliomyelitis. Consequently, year after year, new concepts are presented and old ones discarded that are intended to show where the virus originates outside the body and to establish its portal of entry into the body. Clearly evident are the facts that there is more to indicate that the virus of poliomyelitis is of endogenous origin.

Whether or not one is of the opinion that a poison or virus, or both, are responsible for human poliomyelitis, it is obvious that the fruit, vegetables, milk, and water used by the poliomyelitis patient prior to his or her illness should be carefully considered. If by

careful inquiry a fruit, vegetable, milk, or water source is found, prevention of other cases of this disease can be brought about by warning the public.

Since a poison or virus will cause damage by disturbing normal chemical relationships within the body, particularly enzyme systems, it is imperative, therefore, to determine what chemical changes take place in human poliomyelitis and how they may be restored to normal. It is at this point that much research can be done. A preventive and therapeutic agent could doubtless be developed to maintain or restore normal chemical relationships within the human body to prevent and cure poliomyelitis. It must be seriously considered that if it were possible with a vaccine to prevent the synthesis or activation of the poliomyelitis virus within the human body, it does not follow that a poisonous or toxic activator would be deprived of its ability to cause neurotoxic damage to the lower motor neurone with resulting paralysis.

SUMMARY

1. The exogenous virus theory of cause of human poliomyelitis fails to explain all facts without exception and cannot be considered to be entirely valid.

2. It is emphasized in this report that the fundamental cause of human poliomyelitis appears to be a poison or toxin and that the virus is synthesized or activated within the human body as a result of the poisoning.

3. There appears to be an intimate relationship between virus diseases and diseases resulting from toxic causes. This fact, illustrated by examples, has been stressed.

4. It is pointed out that the poisonous activating factor in cases of human poliomyelitis can originate from fruits, vegetables, milk, and water during epidemics of this disease.

5. The locality influences, seasonal incidence, simultaneous development of multiple cases in homes, institutions and communities, as well as the visceral lesions and other facts, all indicate the association of a poison or toxin with human poliomyelitis.

6. Normal chemical relationships within the body are disturbed in cases of human poliomyelitis; a preventive and therapeutic agent could doubtless be developed to maintain or restore these relationships and thus prevent and cure the disease.

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A LABORATORY DIAGNOSTIC SIGN FOR PERTUSSIS*

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The textbooks and the current literatures state that the laboratory aids for the diagnosis of pertussis are the cough plate culture, the high white count with a high predomination of lymphocytes, and the agglutination slide test which determines if the antigen response to former pertussis immunization had been sufficient or not.

One hundred and sixteen cases of pertussis were reviewed at the Kern General Hospital during the past five years. Three facts seemed to stand out in the uncomplicated pertussis cases: 1. The usual high white count with the preponderance of lymphocytes occurring in 111 cases. 2. The low incidence of positive cultures for Hemophilus pertussis. (18 positive cough plates out of the 116 cases). 3. The extreme low sedimentation rate of one millimeter in one hour.

It is the latter fact that stimulated this paper. The past four years showed that sedimentation rates were not ordered on the uncomplicated pertussis cases, but when this fact became manifest then routine sedimentation rates were ordered. The last twenty-two cases of pertussis all showed a sedimentation rate of one millimeter. This fact repeats itself, thus it makes diagnosis confirmation much easier even though the first pertussis cough plate culture may be negative. The cases that were suspect pertussis with high sedimentation rates did not fit the picture of uncomplicated pertussis.

When one reviews records, one finds that the control element is usually missing. The obvious repeated fact, such as the low sedimentation rate in these cases, was the clue to repeat this laboratory procedure. In the last sixteen months routine sedimentation rates were ordered on all suspect uncomplicated pertussis cases and out of twenty-two cases, twenty-two gave sedimentation rates of one millimeter. Over ninety per cent of these cases were clinically classic "whoopers." The sedimentation rate over ten usually began to show pneumonia signs confirmed by x-ray and physical examinations.

The disease, in relation to the physico-chemical mechanism in-

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fluencing the red cells or the serum globulin and plasma fibrinogen, is beyond this article in trying to rationalize on the low sedimentation rate. Nevertheless, the fact that low sedimentation rate in uncomplicated pertussis repeatedly manifests itself is worthy enough to make it a must in ordering laboratory work for the diagnosis of pertussis.

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DRAINAGE IN TUBERCULOUS MENINGITIS. (Archives Francaises De Pédiatrie, Paris, 10:785, No. 8, 1953). An improved technique, using a polythene tube, of continuous external ventricular drainage of the cerebrospinal fluid for the relief of intracranial hypertension in tuberculous meningitis has been used by the author and his colleagues to perform 77 drainings in 64 children with tuberculous meningitis. Significant amounts of cerebrospinal fluid—200 to 300 cc. daily—were removed fro meach child. Technical difficulties were reduced to a minimum but could not be eliminated entirely. In this series of 64 patients, 19 were cured and 45 died. In those cured, the hypertension was relieved, as evidenced by a progressive decrease in the amount of fluid being drained. In some patients the amount showed no tendency to decrease. Drainage produced its most spectacular effects—disappearance of headache and vomiting and regression of disturbances in consciousness—on the older children and on the ones in whom the disease had been diagnosed relatively early in its progress. It did not have any significant effects on children who were very young (under 5 years), unconscious, or already definitely comatose. The importance of the drainage method as a factor in the cures effected cannot be evaluated. The fact that many patients remained unconscious despite ventricular drainage shows that intracranial hypertension per se is not as important a cause of critical symptoms as are the neural lesions themselves. For patients in whom the amount of cerebrospinal fluid drained does not significantly decrease within the first 10 days of drainage, a special neurosurgical intervention should be devised (eventually an internal shunt of the cerebrospinal fluid from the meningeal spaces into a cavity of the temporal bone).

—*J.A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

EYE SYMPTOMS OF INFANTILE SCURVY*

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The eye symptoms of infantile scurvy receive small notice in works on ophthalmology, and the writer has recently seen a consultation of eight oculists result in much difference of opinion as to the significance, cause, and treatment of proptosis in a scorbutic baby.

The history of the case is interesting, as it is one of the few where the cause of the protrusion of the eyeball was shown by autopsy to be an extensive subperiosteal hemorrhage of the orbital bones.

The patient was the offspring of healthy young parents, who had two older healthy children. It was born after a normal labor. It showed an intolerance of cow's milk, and was given cereal milk, upon which it throve for nine months, when it developed gradually pain, swelling and pseudoparalysis in both lower extremities. The loss of power was attributed to an obscure spinal injury by the family physician. Some weeks later, a slight protrusion of the left eyeball appeared, associated with blackened lids. Ten days later, a sudden protrusion of the right eyeball occurred, so extreme in degree that the closed lids left a large uncovered space on the cornea; both lids had a dark, bruised-like color, and were much swollen. Four days afterwards the baby became feverish and looked very ill.

The child was seen at 9 P.M., January 18, by Dr. Lorenzo Burrows, who decided that the extreme proptosis of the right eye was not due to sarcoma, orbital cellulitis, or fracture of the skull, and referred the matter to Dr. Irving Snow as a medical case.

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January 19. Examination by Dr. Irving Snow, 9 A.M. Child very apathetic; good development; slight rachitic rosary, heart rapid and weak; nothing in chest or abdomen; temperature, 100°F.

Right eyeball protruding as in the most extreme case of exophthalmic goitre; cornea cloudy; upper and lower eyelids black and swollen, and do not cover the eyeball; no chemosis; eyeball freely movable; no discoloration of conjunctiva.

Moderate proptosis of left eyeball, with swollen, discolored lids; left cornea clear.

Gums slightly swollen and violet colored. No teeth.

Upper extremities, normal.

Legs below knee uniformly swollen and painful. Feet edematous. No purpuric spots or heat of surface.

The child thus presented a typical picture of infantile scurvy. The proptosis and swelling and pseudoparalysis of the legs were due to subperiosteal hemorrhage. Orange juice, fresh milk and stimulants were given, but the temperature rose to 107.5°F. at 3:30 P.M. The child showed increasing prostration, and died at 11 P.M., after a stay of twenty-six hours in the hospital.

Autopsy. An examination of only right eye permitted. A huge hematoma lay between the periosteum and bone of the orbit, filling the pyramidal space behind the eye and extending nearly around the entire orbit, stripping off nearly the whole orbital periosteum. No retinal hemorrhages. A smear from the clot showed large quantities of pus containing a bacillus like the influenza bacillus. Subperiosteal hemorrhage occurred in both orbits —in the left orbit, earlier and slighter in degree, and spontaneously ceasing. In the right orbit, an extensive, sudden hemorrhage took place, pushing forward the eyeball.

The rise of temperature in the last few hours of life was due to some terminal infection, probably proceeding from the infected retrobulbar hematoma.

The question of operative relief of the exophthalmos by removing the clot behind the eyeball was discussed, but was dismissed as unpractical on account of the moribund condition of the patient and the usual spontaneous absorption of the clot in a more favorable case.

Of the collective investigations of the American Pediatric So-

ciety of 340 cases of scurvy, eye symptoms were mentioned in 49; a swelling mentioned in 9; in 18 protrusion of the eyeball; and in 22 both symptoms occurred. In the six autopsies reported by the collective investigation, in none was an examination of the eye made. Heubner reports 65 cases of infantile scurvy, orbit involved four times.

Regarding adult scurvy, Buzzard (Reynolds' System) says, in some cases at an early period: "The integument around one or both orbits is puffed up into a bruise-colored swelling; the conjunctiva covering the sclerotic is turned and of a brilliant red color throughout; the cornea lies at the bottom of a circular trench or well. There is nothing inflammatory about the condition. It resembles a very violent ophthalmia in color, but with no pain or discharge."

Sudden exophthalmos in a baby should at once excite suspicions of scurvy. It may, in fact, be the first indication of the disease. The rapidity with which the proptosis occurs indicates a hemorrhagic lesion.

Barlow (Grancher's System) says of infantile scurvy: "A remarkable symptom in one or both orbits is not rare. Suddenly, without premonitory sign or appreciable cause, we find a moderate exophthalmos, with the ocular globes turned downward. This symptom appears and augments for twenty-four hours. When it has reached its acme, one finds a deep ecchymosis and thickening of the superior lids, due to a hemorrhagic effusion. Both orbits may be affected, but successively and in an unequal manner. The proptosis may be only slight. There is no increase of intraocular tension or hemorrhage into the deeper parts of the eye. The eyeball is freely movable, not anchored by an inflammatory exudation, as in orbital cellulitis. The hemorrhage effusion into the eyelids affects the deeper parts; the conjunctiva of the lid and globe are only occasionally affected.

Similar cases of scorbutic proptosis have been collected, as follows:

Barlow (Keating's System) describes 2 cases of scorbutic proptosis. He says it is dependent on a bone lesion, and extravasation of blood between the orbital plate of the frontal and its subjacent periosteum, the extravasation pushing out the eyeball.

(1) Eight-months-old baby, brought on account of proptosis

of one eyeball. Great tenderness in the limbs, and general cachexia. Since early infancy the baby has been rather tender in the legs. During the last week the tenderness became excessive; both upper eyelids became suddenly swollen three weeks ago, the right upper lid purplish-red color, due to deep extravasation into its substance. No ecchymosis of palpebral of ocular conjunctiva. Slight proptosis of right eye. Cure after antiscorbutics.

(2) Ten-months-old baby, artificially fed. Legs tender at eighth month; at nine months the wrists became tender; there later developed ecchymosis in both eyelids; pseudoparalysis and slight proptosis of left eye.

Child died after an illness of three months. The autopsy showed subperiosteal hemorrhages. Eye not examined.

(3) Nicholai. (Nederlausch. Tijdschrift Vor Geneekunde). Child nine months, brought to Nicholai for a sudden protrusion of the left eye. On both upper eyelids, there was a small greenish-yellow spot. Child otherwise well. Two days after Nicholai's first examination, a neuroparalytic keratitis developed, and Nicholai found in the gums the characteristic changes of scurvy. A few days later a retrobulbar hemorrhage occurred in the right eye. On antiseptic washes and antiscorbutics the keratitis and ocular protrusion disappeared.

(4) Ed. Meyer. Seven-months-old child, previously well, attacked with edematous swelling and pain in left ankle; left-sided exophthalmos. Death with increasing anemia. Autopsy: Rachitis, anemia, hemorrhagic pachymeningitis, hemorrhagic periostitis of the left orbit and tibia, fatty myocarditis, hemorrhages into heart, lungs and kidney.

(5) Ashby and Wright. Girl, seven-months-old, fed on dried milk and maltose-food for seven months. Throve and looked like a prize baby. Bad cough for two to three weeks. Two weeks before examination, the left eyeball became suddenly prominent, and protruded so much that the nurse thought it would drop out. The proptosis was attributed to a slight blow on the eye the baby received from a comforter. The eye had been prominent for two weeks, during which time the baby had several bad fainting attacks. The baby was large and fat and pale; temperature 101°F.; drowsy, but readily aroused. The eyeball was exposed in part;

the lids when closed did not meet; lids not puffy or ecchymosed; ribs beaded; no teeth, gums normal. Urine stained the diaper brown. Rapid improvement on fresh milk and orange juice.

It is interesting to note that so acute and experienced an observer as Dr. Nansen looks on adult scurvy as a disease not due to the absence of a certain element in the food, but to the presence of ptomaines in badly preserved salt beef or other preserved foods. He believes that with sterilized foods and properly given, there would be no scurvy, and that orange and lime juice are unnecessary. Both Dr. Nansen and Dr. Caillé believe that scurvy in adults and children is due to ptomaine poisoning.

REGIONAL LYMPHADENITIS AND "CAT SCRATCH DISEASE." (Medical Journal of Australia, Sydney, 2:81, July 18, 1953). During the last two years nine adults and six children with a peculiar regional nonbacterial suppurative lymphadenitis were studied in Queensland. The clinical behavior of these 15 patients and the microscopic detail of the affected glands presented a striking similarity to the lymphadenitis accurring in patients with "cat scratch disease." Of 10 patients from whom a reasonably complete history was obtained, 6 gave a history of contact with cats, and of these, 3 stated that they had been scratched or bitten not long before the onset of the lymphadenitis. All three patients on whom an intradermal test was carried out with the "cat scratch" antigen supplied from London gave a positive reaction. Thus the authors believe that the condition observed in Queensland is identical with the disease described as "cat scratch disease" in America, England, and France. The disease is thought to be caused by a virus of which the cats may act purely as passive carriers. There was no noticeable response of the patients either to sulfonamides or to penicillin, but recovery occurred without sequelae. Although the disease will follow a benign course in most of the cases, there is evidence to suggest that the condition may not necessarily be always benign or localized in character.—*J.A.M.A.*

DEPARTMENT OF ABSTRACTS

COPAITICH, T. AND ANDREONI, G.: DEMONSTRATION OF KOCH BACILLUS IN SPINAL FLUID BY ELECTROPHORESIS. (Aggionamento Pediatrico, 4:857, Dec. 1953).

The early demonstration of tubercle bacilli in meningitis is a great advantage. The authors were able to demonstrate tubercle bacilli in eleven out of twelve cases in the spinal fluid of meningitic cases by electrophoresis, whereas the culture was only positive in four of the twelve cases. They have noted that during the process of electrophoresis, lymphocytes and albumin accumulate around the anode, whereas polymorphs and the tubercle bacilli accumulate around the cathode. The bacilli are then demonstrated by staining the droplets from the cathode with Ziehl-Neelsen stain.

MICHAEL A. BRESCIA, M.D.

ALLISON, A. C.: PROTECTION AFFORDED BY SICKLE-CELL TRAIT AGAINST SUBTERTIAN MALARIAL INFECTION. (British Medical Journal, 4857:290, Feb. 6, 1954).

A study has been made of the relationship between the sickle-cell trait and subtertian malarial infection. It has been found that in indigenous East Africans the sickle-cell trait affords a considerable degree of protection against subtertian malaria. The incidence of parasitaemia in 43 Ganda children with the sickle-cell trait was significantly lower than in a comparable group of 247 children without the trait. An infection with P. falciparum was established in 14 out of 15 Africans without the sickle-cell trait, whereas in a comparable group of 15 Africans with the trait only two developed parasites. It is concluded that the abnormal erythrocytes of individuals with the sickle-cell trait are less easily parasitized by P. falciparum than are normal erythrocytes. Hence, those who are heterozygous for the sickle-cell gene will have a selective advantage in regions where malaria is hyperendemic. This fact may explain why the sickle-cell gene remains common in these areas in spite of the elimination of genes in patients dying of sickle-cell anemia. The implications of these observations in other branches of haematology are discussed. AUTHOR'S SUMMARY.

LORBER, J.: THE RESULTS OF TREATMENT OF 549 CASES OF TUBERCULOUS MENINGITIS. (American Review of Tuberculosis, 69:13, Jan. 1954).

The author analyzes the results of 549 cases of tuberculous meningitis, between 1947 and 1950, treated by prolonged, combined, intramuscular and intrathecal streptomycin. The over-all two year survival rate of 101 patients admitted in 1947 was 31.6 per cent and of 448 patients admitted 1948-1950 was 49.3 per cent. The most important prognostic factor was the stage of disease on admission. Of 100 patients admitted early in the disease 74 per cent survived, whereas only 24.8 per cent of 125 advanced cases survived. Of 161 patients with both meningitis and miliary tuberculosis, 44.1 per cent survived, whereas 52.2 per cent of 287 patients with only meningitis survived. There was no difference between the survival rates of patients in the age groups 3 to 14 years and more than 14 years; but the prognosis for those less than 3 years of age was worse. This less favorable prognosis in younger children was partly due to delay in diagnosis. The 245 survivors were followed for 2½ to 6 years. In all, 64 per cent had no sequelae, whatever. The incidence of moderate or severe neurologic sequelae was least among early cases (9 per cent) and greatest among late cases (55 per cent). In 14.7 per cent of all survivors,. deafness was the only residual damage. The deafness is presumed to be due to the streptomycin.

MICHAEL A. BRESCIA, M.D.

WALLIS, A. D.: DIETARY EGGS AND RHEUMATIC FEVER. (American Journal Medical Sciences, 227:167, Feb. 1954).

Because eggs are by far the richest source of dietary choline necessary for the synthesis of serum phospholipid, which in turn is an active component of the natural serum inhibitor of the streptococcol hemolysin known as streptolysin S, dietary histories with regard to eggs were taken from 184 patients with rheumatic heart disease and 1380 normal persons. Of the cardiac subjects, 10.2 per cent actively disliked eggs and 40.6 per cent believed they ate few eggs in childhood either from dislike or inaccessibility, against corresponding percentages of 4.6 and 16.3 for the control group. A history of few eggs in childhood was given by 49 per cent of the patients with cardiac damage severe enough to require digi-

talis and by 51 per cent of those who had had three or more acute episodes of rheumatic fever. It is suggested that individual differences not only in choline intake but also in choline requirement may play a part in determining susceptibility to rheumatic fever.
AUTHOR'S SUMMARY.

WHITE, R.H.R. AND STANDEN, O.D.: PIPERAZINE IN THE TREATMENT OF THREADWORMS IN CHILDREN. (British Medical Journal, 4839:755, Oct. 3, 1953).

In clinical trials of piperazine hydrate, gentian violet and diphenan against oxyuriasis in 136 children, piperazine hydrate syrup and gentian violet showed marked oxyricidal properties, while diphenan was inactive. Owing to lack of toxicity and side-effects, to ease of administration, and to its excellent therapeutic properties, piperazine hydrate syrup at a dose level of 50-75mg. per Kg. per day is considered to be the drug of choice for oxyuriasis in children. Of 31 children treated at levels greater than 50 mg. per Kg. daily, 97 per cent were cured. The use of suppositories is unnecessary. Spontaneous cure may be expected in 19 per cent of cases of oxyuriasis. AUTHOR'S SUMMARY.

SHANE, S. J. AND RILEY, C.: TUBERCULOUS MENINGITIS. COMBINED THERAPY WITH CORTISONE AND ANTIMICROBIAL AGENTS. (New England Journal of Medicine, 249:829, Nov. 19, 1953).

Seven cases of tuberculous meningitis treated with combined cortisone and antimicrobial agents, with five recoveries are reported. In one case the resolution of an established subarachnoid block occurred during treatment, and in another, there is evidence that the development of an incipient block was suppressed by cortisone. In one case clinical and bacteriological relapse, which had occurred after neurosurgical treatment (burr holes), was reversed by this form of combined therapy. The theoretical basis for this form of treatment is the known ability of the adrenal steroid to suppress the inflammatory process, or to bring about its reversal. The use of cortisone in the presence of active tuberculosis appears to be a safe procedure, provided that it is adequately covered by effective antimicrobial therapy.
MICHAEL A. BRESCIA, M.D.

BOOK REVIEWS

PEDIATRIC GYNECOLOGY. By Goodrich C. Schauffler, M.D. 3rd Ed., Cloth, Pp. 318. Illustrated. Price $7.50. Chicago: The Year Book Publishers, Inc., 1953.

This book is of great value and contains much information relative to the female genital tract of the infant and child. A specialized book of this nature is an important addition to any physician's library who deals with female infants and children. The text is concise, easily read and the accompanying illustrations in most instances are revealing and informative. The book is highly recommended. MICHAEL A. BRESCIA, M.D.

THE CONCEPTION OF DISEASE. By Walther Riese, M.D. Cloth. Pp. 100. Price $3.75. New York: Philosophical Library, 1953.

This is a text that deals with the concept of disease in a purely philosophical manner. There is an attempt at continuity of thought from the stoic conception of disease at the beginning to the final chapters on disease and health. The final chapters which concern themselves with the more modern views of disease are also treated from a philosophical viewpoint rationalizing their inadequacy and shortcomings. This book does not deal with the concepts of disease in a conventional manner but rather from the viewpoint of the philosopher. H. FRUCHTER, M.D.

THE SURGERY OF INFANCY AND CHILDHOOD. By Robert E. Gross, M.D., D.Sc. Cloth. Pp. 1000. Illustrated. Price $16.00. Philadelphia: W. B. Saunders Company, 1953.

I will state at the outset that this is an excellent book and will make a valuable addition to any physician's library. One can and probably will refer frequently to this volume with profit. The book covers adequately most of the surgical problems which are peculiar to infants and children. Although one might have differences of opinion regarding certain ideas, for example the author begins treatment for undescended tests much later than most other workers would, these criticisms are minor when considering the book as a whole. One might also criticize the use of many x-ray illustra-

tions using contrast media in visualizing intestinal obstructions in infants, even though the author rightly urges restraint in using such media. The pattern of the book is excellent in the proper use of intelligible embryological background in introducing each new subject. The clinical discussions are brief, concise and yet complete enough. The plethora of pictures and illustrations are good and clear.

MICHAEL A. BRESCIA, M.D.

THE JEALOUS CHILD. By Edward Podolsky, M.D. Cloth. Pp. 147. Price $3.75. New York: Philosophical Library, 1954.

This book very simply presents the emotional and behavior problems which beset children in various categories. For example, the special problems which might arise in an only child, a child from a so-called minority group, a child from a family of inferior position than the family of his mates, etc. The best virtue of the book is that it is devoid of the usual psychiatric jargon and clichés. However, the book is superficial and would not lend much interest to the serious student of this subject.

MICHAEL A. BRESCIA, M.D.

TREATMENT OF TETANUS. (British Medical Journal, London, 2:866, Oct. 17, 1953). A 4-year-old girl and a 7-year-old boy with severe tetanus in whom the periods between injury and onset of symptoms were four and six days, i.e., less than one week, died despite treatment with gallamine triethiodide, thiopental sodium, antitoxin, and antibiotics. Death occurred within three days in the girl and within 24 hours in the boy. Gallamine triethiodide was given in doses varying from 5 mg. intravenously to 20 mg. intramuscularly. The girl received a total dose of 180 mg. intramuscularly and 170 mg. intravenously, and the boy received 404 mg. intravenously. The author had the impression that gallamine triethiodide caused some respiratory embarrassment in the girl, but this was not confirmed in the boy, who was, however, desperately ill on admission. Penicillin, even in doses of 1,000,000 units given every four hours, and antitoxin serum in doses of 80,000 units in the first 12 hours had little effect. The present methods of treatment of tetanus in cases occurring within a week of injury are ineffective. Routine active immunization of all children against tetanus is recommended.—*J.A.M.A.*

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